WEST Search History

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DATE: Wednesday, September 20, 2006

Hide?	Set Name	Query	Hit Count
		DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ	
☐	L10	(method$5 or process$5) same L9	17
☐	L9	(BMP? or BMP-1 or (bone with morphogenic with protein))same L8	38
☐	L8	(modulat$5 or inhibit$5 or alter$5 or decreas$5) same L7	199
☐	L7	(promyostatin or myostatin or pro-myostatin or GDF-8)	358

END OF SEARCH HISTORY

=> index bioscience medicine

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS, CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 13:01:52 ON 20 SEP 2006

71 FILES IN THE FILE LIST IN STNINDEX

=> S (promyostatin or myostatin or pro-myostatin or GDF-8)
2 FILE ADISINSIGHT
1 FILE ADISNEWS
70 FILE AGRICOLA
1 FILE ANABSTR
2 FILE ANTE
16 FILE AQUASCI
37 FILE BIOENG
499 FILE BIOSIS
74 FILE BIOTECHABS
74 FILE BIOTECHDS
107 FILE BIOTECHNO
213 FILE CABA
520 FILE CAPLUS
2 FILE CEABA-VTB
10 FILE CIN
30 FILE CONFSCI
3 FILE DDFB
15 FILE DDFU
3685 FILE DGENE
23 FILES SEARCHED...
23 FILE DISSABS
3 FILE DRUGB
20 FILE DRUGU
13 FILE EMBAL
346 FILE EMBASE
269 FILE ESBIOBASE
23 FILE FROSTI
18 FILE FSTA
397 FILE GENBANK
115 FILE IFIPAT
5 FILE IMSDRUGNEWS
5 FILE IMSRESEARCH
16 FILE JICST-EPLUS
102 FILE LIFESCI
346 FILE MEDLINE
1 FILE NTIS
1 FILE NUTRACEUT
5 FILE OCEAN
117 FILE PASCAL
4 FILE PHAR
13 FILE PHIN
42 FILE PROMT
3 FILE PROUSDDR
523 FILE SCISEARCH
95 FILE TOXCENTER
254 FILE USPATFULL
22 FILE USPAT2
3 FILE VETU
65 FILES SEARCHED...
101 FILE WPIDS
2 FILE WPIFV
101 FILE WPINDEX
30 FILE NLDB

51 FILES HAVE ONE OR MORE ANSWERS, 71 FILES SEARCHED IN STNINDEX

L1 QUE (PROMYOSTATIN OR MYOSTATIN OR PRO-MYOSTATIN OR GDF-8)

=> d rank
F1 3685 DGENE
F2 523 SCISEARCH
F3 520 CAPLUS
F4 499 BIOSIS
F5 397 GENBANK
F6 346 EMBASE
F7 346 MEDLINE
F8 269 ESBIOBASE
F9 254 USPATFULL
F10 213 CABA
F11 117 PASCAL
F12 115 IFIPAT
F13 107 BIOTECHNO
F14 102 LIFESCI
F15 101 WPIDS
F16 101 WPINDEX
F17 95 TOXCENTER
F18 74 BIOTECHABS
F19 74 BIOTECHDS

=> file f2-f4, f6-f15

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FILE 'IFIPAT' ENTERED AT 13:04:17 ON 20 SEP 2006
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FILE 'WPIDS' ENTERED AT 13:04:17 ON 20 SEP 2006
COPYRIGHT (C) 2006 THE THOMSON CORPORATION

=> S L1

L2 3512 L1

=> S (modulat? or inhibit? or alter? or decreas? or increas?) (s) L2
6 FILES SEARCHED...
9 FILES SEARCHED...
L3 1855 (MODULAT? OR INHIBIT? OR ALTER? OR DECREAS? OR INCREAS?) (S) L2

=> S (BMP? or BMP-1 or (bone (w) morphogenic (w) protein))(s) L3
10 FILES SEARCHED...
L4 148 (BMP? OR BMP-1 OR (BONE (W) MORPHOGENIC (W) PROTEIN))(S) L3

=> S (method? or process?)(s) L4
8 FILES SEARCHED...
9 FILES SEARCHED...
L5 50 (METHOD? OR PROCESS?)(S) L4

=> dup rem L5
PROCESSING COMPLETED FOR L5
L6 40 DUP REM L5 (10 DUPLICATES REMOVED)

=> d ibib abs L6 1-40

L6 ANSWER 1 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2006:104441 USPATFULL <<LOGINID::20060920>>
TITLE: Use of passive myostatin immunization
INVENTOR(S): El Halawani, Mohamed E., St. Paul, MN, UNITED STATES
You, Seungkwon, Suwon, KOREA, REPUBLIC OF
PATENT ASSIGNEE(S): Regents of the University of Minnesota (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006088543	A1	20060427
APPLICATION INFO.:	US 2005-247691	A1	20051011 (11)

RELATED APPLN. INFO.: Division of Ser. No. US 2001-754826, filed on 4 Jan 2001, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SCHWEGMAN, LUNDBERG, WOESSNER & KLUTH, 1600 TCF TOWER, 121 SOUTH EIGHT STREET, MINNEAPOLIS, MN, 55402, US
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1-8
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 1975
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method to alter the phenotype of animals, e.g., avians, which employs passive and active immunization is provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 2 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2006:80475 USPATFULL <<LOGINID::20060920>>
TITLE: ActRII receptor polypeptides, methods and compositions
INVENTOR(S): Knopf, John, Carlisle, MA, UNITED STATES
Seehra, Jasbir, Lexington, MA, UNITED STATES
PATENT ASSIGNEE(S): Acceleron Pharma Inc., Cambridge, MA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006068468	A1	20060330
APPLICATION INFO.:	US 2005-190202	A1	20050725 (11)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2004-590765P	20040723 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FISH & NEAVE IP GROUP, ROPES & GRAY LLP, ONE

INTERNATIONAL PLACE, BOSTON, MA, 02110-2624, US
NUMBER OF CLAIMS: 35
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 17 Drawing Page(s)
LINE COUNT: 3181
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB In certain aspects, the present invention provides compositions and methods for modulating (promoting or inhibiting) growth of a tissue, such as bone, cartilage, muscle, fat, and/or neuron. The present invention also provides methods of screening compounds that modulate activity of an ActRII protein and/or an ActRII ligand. The compositions and methods provided herein are useful in treating diseases associated with abnormal activity of an ActRII protein and/or an ActRII ligand.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 3 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2006:66928 USPATFULL <<LOGINID::20060920>>
TITLE: Methods to prevent tumor recurrence by blockade of tgf-beta
INVENTOR(S): Berzofsky, Jay A, 5908 BRADLEY BLVD.,, BETHESDA, MD, UNITED STATES 20814-1107
Terabe, Masaki, Bethesda, MD, UNITED STATES
Matsui, So, Ibaraki, JAPAN
PATENT ASSIGNEE(S): GOVERNMENT OF THE UNITED STATES OF AMERICA AS REPRESENTED BY THE SECRETARY, DEPT. OF HEALTH, ROCKVILLE, MD, UNITED STATES, 20852 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006057145	A1	20060316
APPLICATION INFO.:	US 2003-532374	A1	20031024 (10)
	WO 2003-US34023		20031024
			20050421 PCT,371 date

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-421286P	20021025 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: KLARQUIST SPARKMAN, LLP, 121 S.W. SALMON STREET, SUITE #1600, ONE WORLD TRADE CENTER, PORTLAND, OR, 97204-2988, US
NUMBER OF CLAIMS: 35
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Page(s)
LINE COUNT: 2140
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods are provided herein to prevent a tumor recurrence in a subject, involving administering to the subject an agent that blocks the TGF-.beta. signaling pathway. In one embodiment, the agent inhibits the immunosuppressive effects of TGF-.beta.. Also provided is a method of enhancing an immune respond in a subject to inhibit recurrence of a tumor by administering an agent which blocks the TGF-.beta. signaling pathway. A method of enhancing the activity of an immune cell to inhibit recurrence of a tumor by contacting a TGF-.beta. receptor-expressing cell with an agent which blocks the TGF-.beta. signaling pathway is also provided, as are methods of screening for an agent that inhibits or measurably reduces the recurrence of a tumor.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 4 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2006:34746 USPATFULL <<LOGINID::20060920>>
TITLE: GDF3 propeptides and related methods
INVENTOR(S): Knopf, John, Carlisle, MA, UNITED STATES
Seehra, Jasbir, Lexington, MA, UNITED STATES
PATENT ASSIGNEE(S): Acceleron Pharma Inc., Cambridge, MA, UNITED STATES (U.S. corporation)

NUMBER KIND DATE
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PATENT INFORMATION: US 2006030520 A1 20060209
APPLICATION INFO.: US 2005-165963 A1 20050624 (11)

NUMBER DATE
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PRIORITY INFORMATION: US 2004-583073P 20040624 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FISH & NEAVE IP GROUP, ROPES & GRAY LLP, ONE INTERNATIONAL PLACE, BOSTON, MA, 02110-2624, US
NUMBER OF CLAIMS: 50
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 2450
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB In certain aspects, the present invention provides compositions and methods for regulating body weight, in particular, for treating obesity and obesity-associate disorders. The present invention also provides methods of screening compounds that modulate activity of GDF3. The compositions and methods provided herein are also useful in treating diseases associated with abnormal activity of GDF3.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 5 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2006:27952 USPATFULL <<LOGINID::20060920>>
TITLE: BMP10 propeptides and related methods
INVENTOR(S): Seehra, Jasbir, Lexington, MA, UNITED STATES
Knopf, John, Carlisle, MA, UNITED STATES
PATENT ASSIGNEE(S): Acceleron Pharma Inc., Cambridge, MA, UNITED STATES, 02139 (U.S. corporation)

NUMBER KIND DATE
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PATENT INFORMATION: US 2006024783 A1 20060202
APPLICATION INFO.: US 2005-128937 A1 20050512 (11)

NUMBER DATE
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PRIORITY INFORMATION: US 2004-570779P 20040512 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FISH & NEAVE IP GROUP, ROPES & GRAY LLP, ONE INTERNATIONAL PLACE, BOSTON, MA, 02110-2624, US
NUMBER OF CLAIMS: 38
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 2180
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB In certain aspects, the present invention provides BMP10 propeptides for use in treating a variety of disorders including heart disorders and other disorders associated with unwanted activity of the mature BMP10 polypeptide. The present invention also provides methods of screening compounds that modulate activity of BMP10.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 6 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2006:9997 USPATFULL <<LOGINID::20060920>>
TITLE: Antagonism of TGF-beta superfamily receptor signaling
INVENTOR(S): Vale, Wylie, La Jolla, CA, UNITED STATES
Harrison, Craig, Nunawading, AUSTRALIA
Gray, Peter, San Diego, CA, UNITED STATES
Fischer, Wolfgang, Encinitas, CA, UNITED STATES
Choe, Senyon, Solana Beach, CA, UNITED STATES

NUMBER KIND DATE
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PATENT INFORMATION: US 2006008846 A1 20060112
APPLICATION INFO.: US 2005-115877 A1 20050427 (11)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2004-565594P	20040427 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FULBRIGHT & JAWORSKI, LLP, 1301 MCKINNEY, SUITE 5100, HOUSTON, TX, 77010-3095, US
NUMBER OF CLAIMS: 32
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 1872
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Members of the TGF-.beta. superfamily control many physiologic and pathophysiologic processes in multiple tissues and signal via type II and type I receptor serine kinases. Type II activin receptors are promiscuous and known to bind 12 TGF-.beta. ligands including activins, myostatin, BMPs and nodal. Methods are described for the screening and identification of antagonist for TGF-.beta. superfamily members, in particular activin-A antagonist.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 7 OF 40 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2006-424378 [43] WPIDS
DOC. NO. CPI: C2006-133865
TITLE: New chimeric virus vector, useful for administering a nucleic acid to a cell or subject for treatment of e.g. cancer or heart failure, comprises a chimeric adeno-associated virus (AAV) capsid.
DERWENT CLASS: B04 D16
INVENTOR(S): AGBANDJE-MCKENNA, M; BOWLES, D E; GRIEGER, J; LI, C; RABINOWITZ, J E; SAMULSKI, R J
PATENT ASSIGNEE(S): (UYFL) UNIV FLORIDA RES FOUND INC; (UYNC-N) UNIV NORTH CAROLINA
COUNTRY COUNT: 113
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2006066066	A2	20060622 (200643)*		EN	81

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU LV MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KM KN KP KR KZ LC LK LR LS LT LU LV LY MA MD MG MK MN MW MX MZ NA NG NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SM SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2006066066	A2	WO 2005-US45552	20051215

PRIORITY APPLN. INFO: US 2004-636126P 20041215
AN 2006-424378 [43] WPIDS
AB WO2006066066 A UPAB: 20060706
NOVELTY - A chimeric virus vector comprising a chimeric AAV capsid comprising a selective amino acid insertion following amino acid position 264 in an AAV2 capsid subunit or a corresponding change in a capsid subunit from other AAV; and a nucleic acid comprising an AAV terminal repeat sequence and a heterologous nucleic acid sequence, where the nucleic acid is packaged within the chimeric AAV capsid, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:
(1) a pharmaceutical formulation comprising the chimeric virus vector

in a carrier;
(2) administering a nucleic acid to a cell; and
(3) delivering a nucleic acid to a subject.
ACTIVITY - Muscular-Gen.; Hemostatic; Neuroprotective; Antidiabetic; Cytostatic; Antiarthritic; Cardiant; Anticonvulsant; Antiparkinsonian; Nootropic; Immunosuppressive; Antianemic; Ophthalmological.
No biological data given.
MECHANISM OF ACTION - Gene Therapy.
USE - The chimeric virus vector, the pharmaceutical formulation, and the methods are useful for administering a nucleic acid to a cell, and for delivering a nucleic acid to a subject, where the subject has or is at risk for a disorder selected from muscular dystrophy including Duchenne or Becker muscular dystrophy, hemophilia A, hemophilia B, multiple sclerosis, diabetes mellitus, Gaucher disease, Fabry disease, Pompe disease, cancer, arthritis, muscle wasting, heart disease including congenital heart failure or peripheral artery disease, intimal hyperplasia, a neurological disorder including epilepsy, Huntington's disease, Parkinson's disease or Alzheimer's disease, an autoimmune disease, cystic fibrosis, thalassemia, Hurler's disease, Krabbe's disease, phenylketonuria, Batten's disease, spinal cerebral ataxia, LDL receptor deficiency, hyperammonemia, anemia, a retinal degenerative disorder including macular degeneration, adenosine deaminase deficiency, and cancer including tumor-forming cancers. (All claimed).
Dwg.0/14

L6 ANSWER 8 OF 40 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2006-433163 [44] WPIDS
DOC. NO. CPI: C2006-136145
TITLE: Inducing neovascularization in subject, comprises administering conditioned cell culture medium from population of cells comprising AC133 cells, endothelial precursor cells, multipotent adult progenitor cells or mesenchymal stem cells.
DERWENT CLASS: A96 B04 D16
INVENTOR(S): LAUGHLIN, M J; POMPILI, V
PATENT ASSIGNEE(S): (UYCA-N) UNIV CASE WESTERN RESERVE
COUNTRY COUNT: 113
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2006060779	A2	20060608	(200644)*	EN	99

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU LV MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KM KN KP KR KZ LC LK LR LS LT LU LV LY MA MD MG MK MN MW MX MZ NA NG NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SM SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

US 2006165667 A1 20060727 (200650)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2006060779	A2	WO 2005-US43952	20051205
US 2006165667	A1 Provisional	US 2004-633292P	20041203
		US 2005-295311	20051205

PRIORITY APPLN. INFO: US 2004-633292P 20041203; US 2005-295311 20051205
AN 2006-433163 [44] WPIDS
AB WO2006060779 A UPAB: 20060711
NOVELTY - Inducing (M1) neovascularization in a subject, involves administering a composition comprising conditioned cell culture medium from a first population of cells comprising AC133+ cells, endothelial precursor cells, multipotent adult progenitor cells (MAPCs), mesenchymal stem cells, AC133-CD34-CD73-cells, CD34+ cells or their combinations, to

the subject.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a composition for inducing neovascularization in a subject, comprising conditioned cell culture medium from a first population of cells having AC133+ cells, endothelial precursor cells, MAPCs, mesenchymal stem cells, AC133-CD34-CD73-cells, CD34+ cells or their combinations;

(2) an implantable device (I) comprising the composition that induces neovascularization in a subject;

(3) distributing the composition or (I) for use by health care professionals, involves placing the composition into a package under sterile conditions and distributing the package for use by health care professionals; and

(4) providing (M2) a composition for use by health care professionals for the treatment of a disorder in a subject, involves (a) providing a sample of umbilical cord blood, (b) culturing at least one cell from the umbilical cord blood in a cell culture medium to generate conditioned media, (c) concentrating the protein components of the conditioned media and formulating a pharmaceutical composition which comprises at least one component, (d) packaging the composition under sterile conditions, and (e) distributing the package for use by health care professionals in treating the disorder in the subject.

ACTIVITY - Cerebroprotective; Vasotropic; Nephrotropic; Cardiant; Cardiovascular-Gen; Analgesic; Antiapoptotic; Antiinflammatory; Cytostatic; Immunosuppressant.

MECHANISM OF ACTION - Induces neovascularization; p38 kinase inhibitor; JAK-STAT inhibitors; IL-2 inhibitor; CD44 inhibitor; CD3 inhibitor; CD154 inhibitor; CD28 inhibitor; Antioxidant.

Neovascularization in the mouse hind-limb injury by endothelial precursor cells (EPCs) derived from purified umbilical cord blood CD133+ cells. The CD133+ cells were seed at 50000-70000 cell/well in 96-well plates under the endothelial-driving culture conditions. After 7 days of culture, cells were injected intracardially into mice that had undergone hind-limb femoral artery ligation by the specific method. Cell yields ranged from 58-130% of plated CD133+ cells, or 0.26% of the initial number of MNC. Blood flow was measured by laser Doppler flowmeter over time, and the results were expressed as the ratio between the blood flow in the injured and the uninjured leg over time. The results showed increased blood flow in the mouse receiving CD133+ cells 14 days after surgery, when compared to the saline control injected on the same day.

USE - (M1) is useful for inducing neovascularization in a subject. The mammal is afflicted with ischemia chosen from limb ischemia, ischemic cardiomyopathy, myocardial ischemia, cerebrovascular ischemia, renal ischemia, pulmonary ischemia and intestinal ischemia (claimed).

ADVANTAGE - The composition efficiently induces neovascularization in a subject.

DESCRIPTION OF DRAWING(S) - The figure shows a graph illustrating the ELISA analysis of transforming growth factor beta 1 in monoculture or co-culture human mesenchymal stem cells and human umbilical vein endothelial cells.

Dwg.15/23

L6 ANSWER 9 OF 40 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2006-125941 [13] WPIDS
DOC. NO. CPI: C2006-044141
TITLE: New pharmaceutical preparation comprises soluble ActRII polypeptide, useful for treating an ActRII-associated disorder, e.g. muscle loss or insufficient muscle growth, neurodegeneration, or abnormal cell growth and differentiation.
DERWENT CLASS: B04 D16
INVENTOR(S): KNOPF, J; SEEHRA, J
PATENT ASSIGNEE(S): (ACCE-N) ACCELERON PHARMA INC
COUNTRY COUNT: 111
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2006012627	A2	20060202	(200613)*	EN	90

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT

KE LS LT LU LV MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KM KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NG NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SM SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW
US 2006068468 A1 20060330 (200624)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2006012627	A2	WO 2005-US26368	20050725
US 2006068468	A1 Provisional	US 2004-590765P	20040723
		US 2005-190202	20050725

PRIORITY APPLN. INFO: US 2004-590765P 20040723; US 2005-190202 20050725
AN 2006-125941 [13] WPIDS
AB WO2006012627 A UPAB: 20060224
NOVELTY - A pharmaceutical preparation for treating an ActRII-associated disorder comprises a soluble ActRII polypeptide, and a pharmaceutical carrier, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:
(1) a packaged pharmaceutical comprising the pharmaceutical preparation and labeled for use in promoting growth of a tissue or diminishing or preventing loss of a tissue in a human or non-human, where the tissue is selected from bone, cartilage, muscle, fat, or neuron;
(2) a stabilized ActRII polypeptide comprising a soluble ActRII polypeptide and a second portion comprising a stabilizing domain;
(3) an isolated polynucleotide comprising:
(a) a coding sequence for a soluble ActRII polypeptide;
(b) a sequence encoding an ActRII polypeptide, a stop codon, and a sequence that is at least 90% identical to a sequence encoding an ActRII polypeptide; or
(c) a polynucleotide sequence selected from 2 fully defined 1542 or 1539 bp sequences (SEQ ID NO. 7 and 8), the isolated polynucleotide further comprising a non-natural transcription termination codon at least six hundred nucleotides before the 3'-terminus;
(4) a recombinant polynucleotide comprising a promoter sequence operably linked to the polynucleotide;
(5) a cell transformed with the recombinant polynucleotide;
(6) a method of making a soluble ActRII polypeptide;
(7) a method for treating a subject having a disorder associated with muscle loss or insufficient muscle growth;
(8) a method for treating a subject having a disorder associated with neurodegeneration;
(9) a method for treating a subject having a disorder associated with abnormal cell growth and differentiation;
(10) a method for increasing growth of a tissue or decreasing loss of a tissue in a subject;
(11) a method for decreasing the body fat content or reducing the rate of increase in body fat content in a subject;
(12) a method for treating a disorder associated with undesirable body weight gain in a subject;
(13) a method of identifying an agent that stimulates growth of a tissue selected from bone, cartilage, muscle, fat, or neuron;
(14) a method of antagonizing activity of an ActRII polypeptide in a cell;
(15) a method of antagonizing activity of an ActRII ligand in a cell;
(16) a GDF8 antagonist comprising an altered GDF8-binding domain of an ActRII receptor that includes one or more mutations increasing the selectivity of the binding domain for GDF 8 relative to a GDF 8-binding domain of a wild-type receptor;
(17) a GDF8 antagonist comprising a GDF8-binding domain of an ActRII receptor fused to an Fc domain, where the IgG Fc domain comprises one or more mutations; and
(18) a method for treating a disorder associated with abnormal

activity of GDF8.

ACTIVITY - Muscular-Gen.; Neuroprotective; Anabolic; Eating-Disorders-Gen.; Nootropic; Antiparkinsonian; Antiinflammatory; Antiallergic; Immunosuppressive; Antimicrobial; Cytostatic; Anorectic; Antidiabetic; Cardiovascular-Gen.; Hypotensive; Antiarthritic; Osteopathic; Cerebroprotective; Vasotropic; Respiratory-Gen.; Hepatotropic. No biological data given.

MECHANISM OF ACTION - None given.

USE - The soluble ActRII polypeptide is useful for making a medicament for the treatment of a disorder associated with abnormal amount, development or metabolic activity of bone, cartilage, or muscle tissue, comprising administering to a subject in need an amount of the soluble ActRII polypeptide. It is also useful for making a medicament for the treatment of a disorder associated with undesirable body fat content, comprising administering to a subject in need an amount of the soluble ActRII polypeptide. The soluble ActRII polypeptide is also useful for making a medicament for the treatment of a disorder associated with neurodegeneration, comprising administering to a subject in need an amount of the soluble ActRII polypeptide (all claimed). The pharmaceutical preparation is useful for treating an ActRII-associated disorder. It can be used for treating muscle loss or insufficient muscle growth, muscle atrophy, muscular dystrophy, amyotrophic lateral sclerosis, muscle wasting disorder, cachexia, anorexia, DMD syndrome, BMD syndrome, AIDS wasting syndrome, muscular dystrophies, neuromuscular diseases, motor neuron diseases, diseases of the neuromuscular junction, or inflammatory myopathies; neurodegeneration, e.g. Alzheimer's Disease, Parkinson's Disease, or Huntington's disease; abnormal cell growth and differentiation, e.g. inflammation, allergy, autoimmune diseases, infectious diseases, or tumors; and undesirable body weight gain, including obesity, non-insulin dependent diabetes mellitus (NIDDM), cardiovascular disease, cancer, hypertension, osteoarthritis, stroke, respiratory problems, or gall bladder disease;
Dwg.0/17

L6 ANSWER 10 OF 40 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN

ACCESSION NUMBER: 2006049004 ESBIOBASE <<LOGINID::20060920>>

TITLE: The role of myostatin and bone morphogenetic proteins in muscular disorders

AUTHOR: Tsuchida K.

CORPORATE SOURCE: Dr. K. Tsuchida, Institute for Comprehensive Medical Science (ICMS), Division for Therapies Against Intractable Diseases, Fujita Health University, Toyoake, Aichi 470-1192, Japan.
E-mail: tsuchida@fujita-hu.ac.jp

SOURCE: Expert Opinion on Biological Therapy, (2006), 6/2 (147-154), 59 reference(s)
CODEN: EOBTA2 ISSN: 1471-2598

DOCUMENT TYPE: Journal; General Review

COUNTRY: United Kingdom

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Skeletal muscle is the largest organ in the human body, and plays an important role in body movement and metabolism. Skeletal muscle mass is lost in genetic disorders such as muscular dystrophy, muscle wasting and ageing. Chemicals and proteins that restore muscle mass and function are potential drugs that can improve human health and could be used in the clinic. ***Myostatin*** is a muscle-specific member of the transforming growth factor (TGF)-.beta. superfamily that plays an essential role in the negative regulation of muscle growth. ***Inhibition*** of ***myostatin*** activity is a promising therapeutic ***method*** for restoring muscle mass and strength. Potential ***inhibitors*** of ***myostatin*** include follistatin domain-containing proteins, ***myostatin*** propeptide, ***myostatin*** antibodies and chemical compounds. These ***inhibitors*** could be beneficial for the development of clinical drugs for the treatment of muscular disorders. Bone morphogenetic protein (***BMP***) plays a significant role in the development of neuromuscular architecture and its proper functions. ***Modulation*** of ***BMP*** activity could be beneficial for muscle function in

muscular disorders. This review will describe the current progress in therapy for muscular disorders, emphasising the importance of ***myostatin*** as a drug target. .COPYRGT. 2006 Ashley Publications.

L6 ANSWER 11 OF 40 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 2006135050 ESBIOBASE <<LOGINID::20060920>>
TITLE: Developmental roles of the BMP1/TLD metalloproteinases
AUTHOR: Ge G.; Greenspan D.S.
CORPORATE SOURCE: D.S. Greenspan, Department of Pathology, University of Wisconsin, 1300 University Avenue, Madison, WI 53706, United States.
E-mail: dsgreens@wisc.edu
SOURCE: Birth Defects Research Part C - Embryo Today: Reviews, (2006), 78/1 (47-68), 229 reference(s)
CODEN: BDRPDV ISSN: 1542-975X
DOCUMENT TYPE: Journal; General Review
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The astacin family (M12A) of the metzincin subclan MA(M) of metalloproteinases has been detected in developing and mature individuals of species that range from hydra to humans. Functions of this family of metalloproteinase vary from digestive degradation of polypeptides, to biosynthetic ***processing*** of extracellular proteins, to activation of growth factors. This review will focus on a small subgroup of the astacin family; the bone morphogenetic protein 1 (***BMP1***)/Tolloid (TLD)-like metalloproteinases. In vertebrates, the ***BMP1*** /TLD-like metalloproteinases play key roles in regulating formation of the extracellular matrix (ECM) via biosynthetic ***processing*** of various precursor proteins into mature functional enzymes, structural proteins, and proteins involved in initiating mineralization of the ECM of hard tissues. Roles in ECM formation include: ***processing*** of the C-propeptides of procollagens types I-III, to yield the major fibrous components of vertebrate ECM; proteolytic activation of the enzyme lysyl oxidase, necessary to formation of covalent cross-links in collagen and elastic fibers; ***processing*** of NH .sub.2-terminal globular domains and C-propeptides of types V and XI procollagen chains to yield monomers that are incorporated into and control the diameters of collagen type I and II fibrils, respectively; ***processing*** of precursors for laminin 5 and collagen type VII, both of which are involved in securing epidermis to underlying dermis; and maturation of small leucine-rich proteoglycans. The ***BMP1*** /TLD-related metalloproteinases are also capable of activating the vertebrate transforming growth factor-.beta. (TGF-.beta.)-like "chalones" growth differentiation factor 8 (GDF8, also known as ***myostatin***), and GDF11 (also known as ***BMP11***), involved in negative feedback ***inhibition*** of muscle and neural tissue growth, respectively; by freeing them from noncovalent latent complexes with their cleaved prodomains. ***BMP1*** /TLD-like proteinases also liberate the vertebrate TGF-.beta.-like morphogens ***BMP2*** and 4 and their invertebrate ortholog decapentaplegic, from latent complexes with the vertebrate extracellular antagonist chordin and its invertebrate ortholog short gastrulation (SOG), respectively. The result is formation of the ***BMP*** signaling gradients that form the dorsal-ventral axis in embryogenesis. Thus, ***BMP1*** /TLD-like proteinases appear to be key to regulating and orchestrating formation of the ECM and signaling by various TGF-.beta.-like proteins in morphogenetic and homeostatic events. .COPYRGT. 2006 Wiley-Liss, Inc.

L6 ANSWER 12 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2005:294725 USPATFULL <<LOGINID::20060920>>
TITLE: Transgenic non-human animals expressing a truncated activin type II receptor
INVENTOR(S): Lee, Se-Jin, Baltimore, MD, UNITED STATES
McPherron, Alexandra C., Baltimore, MD, UNITED STATES
PATENT ASSIGNEE(S): THE JOHNS HOPKINS UNIVERSITY SCHOOL OF MEDICINE (U.S. corporation)

NUMBER KIND DATE

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PATENT INFORMATION: US 2005257278 A1 20051117
APPLICATION INFO.: US 2005-51267 A1 20050203 (11)
RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-841730, filed on 24 Apr 2001, GRANTED, Pat. No. US 6891082
Continuation-in-part of Ser. No. US 2000-626896, filed on 27 Jul 2000, GRANTED, Pat. No. US 6656475
Continuation-in-part of Ser. No. US 2000-485046, filed on 5 May 2000, GRANTED, Pat. No. US 6696260 A 371 of International Ser. No. WO 1998-US15598, filed on 28 Jul 1998

NUMBER DATE
---------- --------
PRIORITY INFORMATION: US 1997-54461P 19970801 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Lisa A. Haile, J.D., Ph.D., DLA PIPER RUDNICK GRAY CARY US LLP, Suite 1100, 4365 Executive Drive, San Diego, CA, 92121-2133, US
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: 1-5
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 6935
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a substantially purified growth differentiation factor (GDF) receptor, including a GDF-8 (myostatin) receptor, as well as functional peptide portions thereof. In addition, the invention provides a virtual representation of a GDF receptor or a functional peptide portion thereof. The present invention also provides a method of modulating an effect of myostatin on a cell by contacting the cell with an agent that affects myostatin signal transduction in the cell. In addition, the invention provides a method of ameliorating the severity of a pathologic condition, which is characterized, at least in part, by an abnormal amount, development or metabolic activity of muscle or adipose tissue in a subject, by modulating myostatin signal transduction in a muscle cell or an adipose tissue cell in the subject. The invention also provides a method of modulating the growth of muscle tissue or adipose tissue in a eukaryotic organism by administering an agent that affects myostatin signal transduction to the organism.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 13 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2005:81086 USPATFULL <<LOGINID::20060920>>
TITLE: Cell-based therapies for ischemia
INVENTOR(S): Laughlin, Mary J., Shaker Heights, OH, UNITED STATES
Haynesworth, Stephen, Beachwood, OH, UNITED STATES
Pompili, Vincent, Hudson, OH, UNITED STATES
PATENT ASSIGNEE(S): Case Western Reserve University, Cleveland, OH (U.S. corporation)

NUMBER KIND DATE
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PATENT INFORMATION: US 2005069527 A1 20050331
APPLICATION INFO.: US 2004-875643 A1 20040624 (10)
RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-730549, filed on 5 Dec 2003, PENDING

NUMBER DATE
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PRIORITY INFORMATION: US 2002-431347P 20021205 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FISH & NEAVE IP GROUP, ROPES & GRAY LLP, ONE INTERNATIONAL PLACE, BOSTON, MA, 02110-2624
NUMBER OF CLAIMS: 55
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 2390

AB The invention provides, among other things, methods for treating an ischemic tissue in a subject in need thereof. The invention further provides methods for increasing the blood flow to an ischemic tissue in a subject in need thereof, such as to ischemic myocardium. The invention further provides cell-based formulations and related kits.

L6 ANSWER 14 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2005:50433 USPATFULL <<LOGINID::20060920>>
TITLE: Metalloprotease activation of myostatin, and methods of modulating myostatin activity
INVENTOR(S): Lee, Se-Jin, Baltimore, MD, UNITED STATES
McPherron, Alexandra C., Baltimore, MD, UNITED STATES
Greenspan, Daniel S., Madison, WI, UNITED STATES
Pappano, William N., Columbia, MD, UNITED STATES
Wolfman, Neil, Dover, MA, UNITED STATES
Tomkinson, Kathy, Cambridge, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005043232	A1	20050224
APPLICATION INFO.:	US 2003-665374	A1	20030916 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-486863P	20030710 (60)
	US 2003-439164P	20030109 (60)
	US 2002-411133P	20020916 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GRAY CARY WARE & FREIDENRICH LLP, 4365 EXECUTIVE DRIVE, SUITE 1100, SAN DIEGO, CA, 92121-2133
NUMBER OF CLAIMS: 66
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Page(s)
LINE COUNT: 2732
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB It has been determined that metalloprotease cleavage of a myostatin pro peptide results in activation of a latent inactive myostatin to an active form. Accordingly, methods of identifying agents that modulate metalloprotease mediated activation of myostatin are provided, as are agents identified using such methods. Also provided are methods of modulating muscle growth in an organism by increasing or decreasing metalloprotease mediated cleavage of a myostatin pro peptide.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 15 OF 40 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2006-020434 [02] WPIDS
DOC. NO. CPI: C2006-006116
TITLE: New pharmaceutical preparation comprising Cerberus/Coco derivatives, useful for treating muscular dystrophy, motor neuron disease, inflammatory myopathy, a disease of the neuromuscular junction, or metabolic disease.
DERWENT CLASS: B04 D16
INVENTOR(S): KNOPF, J; SEEHRA, J
PATENT ASSIGNEE(S): (ACCE-N) ACCELERON PHARMA INC
COUNTRY COUNT: 111
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2005115439	A2	20051208	(200602)*	EN	52

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KM KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NG NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SM SY TJ TM TN TR TT

TZ UA UG US UZ VC VN YU ZA ZM ZW
US 2006025340 A1 20060202 (200610)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2005115439	A2	WO 2005-US18928	20050527
US 2006025340	A1 Provisional	US 2004-575062P	20040527
		US 2005-140024	20050527

PRIORITY APPLN. INFO: US 2004-575062P 20040527; US 2005-140024 20050527
AN 2006-020434 [02] WPIDS
AB WO2005115439 A UPAB: 20060106
NOVELTY - A pharmaceutical preparation comprising a myostatin antagonist protein including a myostatin binding domain of a Cerberus/Dan/Gremlin polypeptide or its variant, which myostatin antagonist protein binds to and neutralizes one or more of nodal and/or myostatin, where the pharmaceutical preparation is substantially free of pyrogenic materials for administration as a human or veterinarian therapeutic, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) a method for inhibiting myostatin signal transduction in a muscle cell or an adipose tissue cell in an animal;

(2) a method for inducing adipogenic differentiation in an animal;

(3) a method for promoting growth of muscle tissue in an animal;

(4) a method for treating or preventing congestive heart failure;

(5) a method for reducing frailty associated with aging;

(6) a method for increasing bone density or accelerating bone fracture repair in a subject;

(7) a method for attenuating protein catabolic response in a subject;

(8) a method for treating or reducing the severity of a muscular dystrophy, a motor neuron disease, an inflammatory myopathy, a disease of the neuromuscular junction, a myopathy due to endocrine abnormalities, a disease of the peripheral nerve, or a metabolic disease in a patient; and

(9) a method for reducing the severity of a pathologic condition which is characterized, at least in part, by an abnormal amount, development or metabolic activity of muscle or adipose tissue in a subject.

ACTIVITY - Cardiovascular-Gen; Muscular-Gen; Neuroprotective; Antiinflammatory; Immunosuppressive; Endocrine-Gen; Anorectic; Antidiabetic; Anabolic; Eating-Disorders-Gen; Anti-HIV.

No biological data given.

MECHANISM OF ACTION - Myostatin-Antagonist.Q10

USE - The myostatin antagonist protein is useful for preparing a medicament for promoting growth of muscle tissue in a human patient or a non-human mammal (claimed). The preparation and methods are useful for inhibiting myostatin signal transduction in a muscle cell or an adipose tissue cell in an animal, for inducing adipogenic differentiation in an animal, for promoting growth of muscle tissue in an animal, for treating or preventing congestive heart failure, for reducing frailty associated with aging, for increasing bone density or accelerating bone fracture repair in a subject, for attenuating protein catabolic response in a subject, and for reducing the severity of a pathologic condition which is characterized, at least in part, by an abnormal amount, development or metabolic activity of muscle or adipose tissue in a subject. The preparation and methods are useful for treating or reducing the severity of muscular dystrophy (e.g. Duchenne Muscular Dystrophy (DMD), Becker Muscular Dystrophy (BMD), Emery-Dreifuss Muscular Dystrophy (EDMD), Limb-Girdle Muscular Dystrophy (LGMD), Facioscapulohumeral Muscular Dystrophy (FSH or FSHD) (Also known as Landouzy-Dejerine), Myotonie Dystrophy (MMD) (Also known as Steinert's Disease), Oculopharyngeal Muscular Dystrophy (OPMD), Distal Muscular Dystrophy (DD), Congenital Muscular Dystrophy (CMD), Myotonia Congenita (MC), Paramyotonia Congenita (PC), Central Core Disease (CCD), Nemaline Myopathy (NM), Myotubular Myopathy (MTM or MM),or Periodic Paralysis (PP)), motor neuron disease (e.g. myotrophic Lateral Sclerosis (ALS) (Also known as Lou Gehrig's Disease), Infantile Progressive Spinal Muscular Atrophy (SMA, SMAI or WH) (Also known as SMA Type 1, Werdnig-Hoffman), Intermediate Spinal Muscular

Atrophy (SMA or SMA2) (Also known as SMA Type 2), Juvenile Spinal Muscular Atrophy(SMA, SMA3 or KW) (Also known as SMA Type 3, Kugelberg-Welander), Spinal Bulbar Muscular Atrophy (SBMA) (Also known as Kennedy's Disease and X-Linked SBMA), or Adult Spinal Muscular Atrophy (SMA)), inflammatory myopathy (e.g. Dermatomyositis (PM/DM), Polymyositis (PM/DM), or Inclusion Body Myositis (IBM)), disease of the neuromuscular junction (e.g. Myasthenia Gravis (MG), Lambert-Eaton Syndrome (LES), or Congenital Myasthenic Syndrome (CMS)), myopathy due to endocrine abnormalities (e.g. Hyperthyroid Myopathy (HYPTM), or Hypothyroid Myopathy (HYPOTM)), disease of peripheral nerve (e.g. Charcot-Marie-Tooth Disease (CMT), Dejerine-Sottas Disease (DS), or Friedreich's Ataxia (FA)), metabolic disease (e.g. Phosphorylase Deficiency (MPD or PYGM), Acid Maltase Deficiency (AMD), Phosphofructokinase Deficiency (PFKM), Debrancher Enzyme Deficiency (DBD), Mitochondrial Myopathy (MITO), Carnitine Deficiency (CD), Carnitine Palmityl Transferase Deficiency (CPT), Phosphoglycerate Kinase Deficiency (PGK), Phosphoglycerate Mutase Deficiency (PGAM or PGAMM), Lactate Dehydrogenase Deficiency (LDHA), Myoadenylate Deaminase Deficiency (MAD), obesity, or type II diabetes), or wasting disorder (e.g. age-related wasting, cachexia, anorexia, DMD syndrome, BMD syndrome, AIDS wasting syndrome, muscular dystrophies, or neuromuscular diseases).
Dwg.0/8

L6 ANSWER 16 OF 40 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2005-786761 [80] WPIDS
DOC. NO. CPI: C2005-242209
TITLE: Inducing a progenitor cell to proliferate or differentiate, useful for inducing tissue formation, repairing a tissue defect or regenerating tissue by contacting a progenitor cell with a nucleic acid encoding a morphogenic protein or MPSF.
DERWENT CLASS: B04 D16
INVENTOR(S): LEE, J C; YEH, L C
PATENT ASSIGNEE(S): (STYC) STRYKER CORP
COUNTRY COUNT: 111
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2005111069	A2	20051124 (200580)*		EN	87

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KM KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NG NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SM SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2005111069	A2	WO 2005-US16426	20050511

PRIORITY APPLN. INFO: US 2004-570388P 20040511
AN 2005-786761 [80] WPIDS
AB WO2005111069 A UPAB: 20051213
NOVELTY - Inducing a progenitor cell to proliferate or differentiate comprises contacting a progenitor cell with a nucleic acid encoding a morphogenic protein and a nucleic acid encoding a morphogenic protein stimulatory factor (MPSF).
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for:
(1) a method for inducing tissue formation, repairing a tissue defect or regenerating tissue, at a target locus in a mammal, comprising administering to the target locus a nucleic acid encoding a morphogenic protein and a nucleic acid encoding a MPSF; and
(2) a method of inducing tissue formation, repairing a tissue defect or regenerating tissue, by in vivo gene therapy, comprising administering to target locus in a patient, a viral vector comprising a nucleotide

sequence that encodes a morphogenic protein and a viral vector comprising a nucleotide sequence that encodes a MPSF so that the morphogenic protein and MPSF are expressed from the nucleotide sequence in the mammal in an amount sufficient to induce progenitor cells to proliferate or differentiate.

USE - The method is useful for inducing tissue formation, repairing a tissue defect or regenerating tissue (claimed).

Dwg.0/10

L6 ANSWER 17 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2004:334890 USPATFULL <<LOGINID::20060920>>
TITLE: Cystine knot growth factor mutants
INVENTOR(S): Weintraub, Bruce D., Rockville, MD, UNITED STATES
Szkudlinski, Mariusz W., Potomac, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004265972	A1	20041230
APPLICATION INFO.:	US 2004-826324	A1	20040419 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-813398, filed on 20 Mar 2001, PENDING Continuation of Ser. No. WO 1999-US5908, filed on 19 Mar 1999, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	WO 1998-US19772	19980922

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: PIPER RUDNICK LLP, Supervisor, Patent Prosecution Services, 1200 Nineteenth Street, N.W., Washington, DC, 20036-2412
NUMBER OF CLAIMS: 198
EXEMPLARY CLAIM: CLM-01-19
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 14192
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions and methods based on mutant Cystine Knot Growth Factors (CKGFs) comprising amino acid substitutions relative to the wild type hormone/growth factor. Mutated glycoprotein hormones, including thyroid stimulating hormone (TSH) and chorionic gonadotropin (CG) are disclosed as exemplary mutant CKGFs. Mutant TSH heterodimers and hCH heterodimers possessed modified bioactivities, including superagonist activity. Accordingly, the present invention provides methods for using mutant CKGFs, CKGF analogs, fragments, and derivatives thereof for treating or preventing diseases. Pharmaceutical and diagnostic compositions, methods of using mutant TSH heterodimers and TSH analogs with utility for treatment and prevention of metabolic and reproductive diseases are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 18 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2004:326851 USPATFULL <<LOGINID::20060920>>
TITLE: Cell-based therapies for ischemia
INVENTOR(S): Laughlin, Mary J., Shaker Heights, OH, UNITED STATES
Haynesworth, Stephen, Beachwood, OH, UNITED STATES
Pompili, Vincent, Hudson, OH, UNITED STATES
PATENT ASSIGNEE(S): Case Western Reserve University, Cleveland, OH, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004258670	A1	20041223
APPLICATION INFO.:	US 2003-730549	A1	20031205 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-431347P	20021205 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: ROPES & GRAY LLP, ONE INTERNATIONAL PLACE, BOSTON, MA, 02110-2624
NUMBER OF CLAIMS: 61
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 18 Drawing Page(s)
LINE COUNT: 1964
AB The invention provides, among other things, methods for treating an ischemic tissue in a subject in need thereof. The invention further provides methods for increasing the blood flow to an ischemic tissue in a subject in need thereof, such as to ischemic myocardium. The invention further provides cell-based formulations.

L6 ANSWER 19 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2004:285806 USPATFULL <<LOGINID::20060920>>
TITLE: ActRIIB fusion polypeptides and uses therefor
INVENTOR(S): Wolfman, Neil M., Dover, MA, UNITED STATES
Bouxsein, Mary L., Brookline, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004223966	A1	20041111
APPLICATION INFO.:	US 2003-689677	A1	20031022 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-421041P	20021025 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Finnegan, Henderson, Farabow,, Garrett & Dunner, L.L.P., 1300 I Street, N.W., Washington, DC, 20005-3315
NUMBER OF CLAIMS: 28
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 1798
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods and compositions for inhibiting growth and differentiation factor-8 (GDF-8) activity in vitro and in vivo are provided. The methods and composition can be used for diagnosing, preventing, or treating degenerative disorders of muscle, bone, or glucose homeostasis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 20 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2004:184499 USPATFULL <<LOGINID::20060920>>
TITLE: Neutralizing antibodies against GDF-8 and uses therefor
INVENTOR(S): Veldman, Geertruida M., Sudbury, MA, UNITED STATES
Davies, Monique V., Harpswell, ME, UNITED STATES
Song, Kening, Arlington, MA, UNITED STATES
Wolfman, Neil M., Dover, MA, UNITED STATES
Bridges, Kristie Grove, Maynard, MA, UNITED STATES
Field, Anne, Royston, UNITED KINGDOM
Russell, Caroline, Royston, UNITED KINGDOM
Valge-Archer, Viia, Little Abington, UNITED KINGDOM

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004142382	A1	20040722
APPLICATION INFO.:	US 2003-688925	A1	20031021 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-419964P	20021022 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Finnegan, Henderson, Farabow,, Garrett & Dunner, L.L.P., 1300 I Street, N.W., Washington, DC, 20005-3315
NUMBER OF CLAIMS: 42
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 18 Drawing Page(s)

LINE COUNT: 1786
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The disclosure provides novel antibodies against growth and differentiation factor-8 (GDF-8), in particular human antibodies, and antibody fragments, including those that inhibit GDF-8 activity in vitro and/or in vivo. The disclosure also provides methods for diagnosing, preventing, or treating degenerative disorders of muscle or bone, or disorders of insulin metabolism.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 21 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2004:178948 USPATFULL <<LOGINID::20060920>>
TITLE: Metalloprotease activation of myostatin, and methods of modulating myostatin activity
INVENTOR(S): Wolfman, Neil, Dover, MA, UNITED STATES
Tomkinson, Kathy, Cambridge, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004138118	A1	20040715
APPLICATION INFO.:	US 2003-662438	A1	20030916 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-486863P	20030710 (60)
	US 2003-439164P	20030109 (60)
	US 2002-411133P	20020916 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FINNEGAN, HENDERSON, FARABOW,, GARRETT & DUNNER, L.L.P., 1300 I Street, N.W., Washington, DC, 20005
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Page(s)
LINE COUNT: 2598
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB It has been determined that metalloprotease cleavage of a myostatin pro peptide results in activation of a latent inactive myostatin to an active form. Accordingly, methods of identifying agents that modulate metalloprotease mediated activation of myostatin are provided, as are agents identified using such methods. Also provided are methods of modulating muscle growth in an organism by increasing or decreasing metalloprotease mediated cleavage of a myostatin pro peptide.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 22 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2004:161274 USPATFULL <<LOGINID::20060920>>
TITLE: Functionalized TGF-.beta. fusion proteins
INVENTOR(S): Wolfraim, Lawrence A., Silver Spring, MD, United States
Letterio, John J., Bethesda, MD, United States
PATENT ASSIGNEE(S): The United States of America as represented by the Secretary of the Department of Health & Human Services, Washington, DC, United States (U.S. government)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6756215	B1	20040629
APPLICATION INFO.:	US 2001-17372		20011019 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-242292P	20001020 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Kemmerer, Elizabeth
ASSISTANT EXAMINER: Nichols, Christopher James
LEGAL REPRESENTATIVE: Klarquist Sparkman, LLP
NUMBER OF CLAIMS: 24

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Figure(s); 7 Drawing Page(s)
LINE COUNT: 4472
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This disclosure relates to TGF-.beta. family protein fusions that display substantial native TGF-.beta. family protein function while also having an additional functionality conveyed by the addition of a functionalizing peptide domain. Such functionalizing peptide domain can be a tag peptide (e.g., an epitope tag, a purification tag, a molecular size differentiation tag, etc.) or a passenger or targeting protein. Also provided are methods of making these fusions, as well as methods of using them for diagnosis and diagnosis of various conditions, in measuring and monitoring levels of the fusion molecule in experimental systems and subjects, and in measuring and detecting receptor proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 23 OF 40 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2004-270031 [25] WPIDS
CROSS REFERENCE: 2004-269880 [25]
DOC. NO. CPI: C2004-105096
TITLE: Modulating myostatin activation, useful for treating metabolic disorders, comprises contacting a latent myostatin complex and a metalloprotease, with an agent that increases or decreases proteolytic cleavage of the pro-peptide.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): GREENSPAN, D S; LEE, S; MCPHERRON, A C; PAPPANO, W N; TOMKINSON, K; WOLFMAN, N; WOLFMAN, N M; TOMKINSON, N W K
PATENT ASSIGNEE(S): (UYJO) UNIV JOHNS HOPKINS; (GREE-I) GREENSPAN D S; (LEES-I) LEE S; (MCPH-I) MCPHERRON A C; (PAPP-I) PAPPANO W N; (TOMK-I) TOMKINSON K; (WOLF-I) WOLFMAN N; (WISC) WISCONSIN ALUMNI RES FOUND; (AMHP) WYETH
COUNTRY COUNT: 107
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004024890	A2	20040325 (200425)*		EN	92
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
AU 2003267246	A1	20040430 (200462)			
US 2005043232	A1	20050224 (200515)			
BR 2003014270	A	20050802 (200553)			
EP 1578928	A2	20050928 (200563)		EN	
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR					
MX 2005002968	A1	20050901 (200617)			
JP 2006517525	W	20060727 (200650)			42

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004024890	A2	WO 2003-US29079	20030916
AU 2003267246	A1	AU 2003-267246	20030916
US 2005043232	A1 Provisional	US 2002-411133P	20020916
	Provisional	US 2003-439164P	20030109
	Provisional	US 2003-486863P	20030710
		US 2003-665374	20030916
BR 2003014270	A	BR 2003-14270	20030916
		WO 2003-US29079	20030916
EP 1578928	A2	EP 2003-749716	20030916
		WO 2003-US29079	20030916
MX 2005002968	A1	WO 2003-US29079	20030916

MX 2005-2968 20050316
JP 2006517525 W WO 2003-US29079 20030916
JP 2004-572010 20030916

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003267246	A1 Based on	WO 2004024890
BR 2003014270	A Based on	WO 2004024890
EP 1578928	A2 Based on	WO 2004024890
MX 2005002968	A1 Based on	WO 2004024890
JP 2006517525	W Based on	WO 2004024890

PRIORITY APPLN. INFO: US 2003-486863P 20030710; US
2002-411133P 20020916; US
2003-439164P 20030109; US
2003-665374 20030916
AN 2004-270031 [25] WPIDS
CR 2004-269880 [25]
AB WO2004024890 A UPAB: 20060804
NOVELTY - Modulating myostatin activation comprises contacting a latent myostatin complex comprising a myostatin pro-peptide and a myostatin C-terminal fragment, and a metalloprotease that can cleave the myostatin pro-peptide, with an agent that increases or decreases proteolytic cleavage of the pro-peptide by the metalloprotease, thus modulating myostatin activation.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:
(1) a method for increasing muscle mass in a subject by administering to the subject an agent that reduces or inhibits proteolytic cleavage of myostatin pro-peptide by a protease, thus preventing activation of latent myostatin and increasing muscle mass in the subject;
(2) a method of ameliorating a metabolic disorder in subject by administering to a subject an agent that induces or inhibits the proteolytic cleavage of myostatin pro-peptide by a protease, thus preventing activation of latent myostatin and ameliorating the metabolic disorder;
(3) a method of identifying an agent that modulates metalloprotease mediated activation of latent myostatin; and
(4) an agent as identified above, or that modulates metalloprotease-mediated activation of a latent myostatin.
ACTIVITY - Metabolic-Gen; Cytostatic; Immunosuppressive; Anorectic; Antidiabetic.
No biological data given.
MECHANISM OF ACTION - Metalloprotease modulator.
USE - The methods and agent are useful for ameliorating a metabolic disorder, e.g. a muscle wasting disorder that is associated with muscular dystrophy, cachexia (associated with cancer or acquired immunodeficiency disease), obesity or type II diabetes (claimed).
Dwg.0/5

L6 ANSWER 24 OF 40 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2004-269880 [25] WPIDS
CROSS REFERENCE: 2004-270031 [25]
DOC. NO. CPI: C2004-105006
TITLE: New agent that modulates metalloprotease-mediated activation of latent myostatin comprising a peptide having a peptide portion of a myostatin polypeptide, useful for treating metabolic disorders, e.g., diabetes.
DERWENT CLASS: B04 D16
INVENTOR(S): TOMKINSON, K; WOLFMAN, N; WOLFMAN, N M
PATENT ASSIGNEE(S): (AMHP) WYETH; (AMHP) WYETH INC; (WYET-I) WYETH; (TOMK-I) TOMKINSON K; (WOLF-I) WOLFMAN N
COUNTRY COUNT: 107
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004024092	A2	20040325 (200425)*		EN	95

RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS
LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP
KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG
PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ
VC VN YU ZA ZM ZW
US 2004138118 A1 20040715 (200447)
AU 2003272394 A1 20040430 (200462)
EP 1549747 A2 20050706 (200544) EN
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV
MC MK NL PT RO SE SI SK TR
BR 2003014380 A 20050519 (200549)
IN 2005000432 P2 20051202 (200604) EN
MX 2005002648 A1 20050901 (200617)
CN 1694957 A 20051109 (200618)
JP 2006507356 W 20060302 (200621) 41

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004024092	A2	WO 2003-US28907	20030916
US 2004138118	A1 Provisional	US 2002-411133P	20020916
	Provisional	US 2003-439164P	20030109
	Provisional	US 2003-486863P	20030710
		US 2003-662438	20030916
AU 2003272394	A1	AU 2003-272394	20030916
EP 1549747	A2	EP 2003-754574	20030916
		WO 2003-US28907	20030916
BR 2003014380	A	BR 2003-14380	20030916
		WO 2003-US28907	20030916
IN 2005000432	P2	WO 2003-US28907	20030916
		IN 2005-KN432	20050315
MX 2005002648	A1	WO 2003-US28907	20030916
		MX 2005-2648	20050309
CN 1694957	A	CN 2003-825168	20030916
JP 2006507356	W	WO 2003-US28907	20030916
		JP 2004-572009	20030916

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003272394	A1 Based on	WO 2004024092
EP 1549747	A2 Based on	WO 2004024092
BR 2003014380	A Based on	WO 2004024092
MX 2005002648	A1 Based on	WO 2004024092
JP 2006507356	W Based on	WO 2004024092

PRIORITY APPLN. INFO: US 2003-486863P 20030710; US
2002-411133P 20020916; US
2003-439164P 20030109; US
2003-662438 20030916
AN 2004-269880 [25] WPIDS
CR 2004-270031 [25]
AB WO2004024092 A UPAB: 20060328
NOVELTY - A new agent that modulates metalloprotease-mediated activation of latent myostatin comprises a peptide comprising a peptide portion of a myostatin polypeptide, or its derivative comprising a peptide having a mutation of a cleavage site for the metalloprotease.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) a method of increasing muscle mass in a subject; and
(2) a method of treating a metabolic disorder in a subject.
ACTIVITY - Antidiabetic; Anorectic; Muscular-Gen; Immunomodulator.
No biological data given.
MECHANISM OF ACTION - Gene therapy.
USE - The agent is useful for preparing a composition for treating metabolic disorders, e.g., diabetes, metabolic disorder associated with

obesity or muscle wasting disorder associated with muscular dystrophy, including Duchenne muscular dystrophy; cachexia, including cachexia associated with cancer or acquired immune deficiency syndrome; or sarcopenia, including age-related sarcopenia.
Dwg.0/5

L6 ANSWER 25 OF 40 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 2004177855 ESBIOBASE <<LOGINID::20060920>>
TITLE: An activin mutant with disrupted ALK4 binding blocks signaling via type II receptors
AUTHOR: Harrison C.A.; Gray P.C.; Fischer W.H.; Donaldson C.; Choe S.; Vale W.
CORPORATE SOURCE: W. Vale, Clayton Found. Labs. Peptide Biol., Salk Institute, San Diego, CA 92037, United States.
E-mail: vale@salk.edu
SOURCE: Journal of Biological Chemistry, (02 JUL 2004), 279/27 (28036-28044), 39 reference(s)
CODEN: JBCHA3 ISSN: 0021-9258
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Activins control many physiologic and pathophysiologic ***processes*** in multiple tissues and, like other TGF-.beta. superfamily members, signal via type II (ActRII/IIB) and type I (ALK4) receptor serine kinases. ActRII/IIB are promiscuous receptors known to bind at least a dozen TGF-.beta. superfamily ligands including activins, ***myostatin*** , several ***BMPs*** , and nodal. Here we utilize a new screening procedure to rapidly identify activin-A mutants with loss of signaling activity. Our goal was to identify activin-A mutants able to bind ActRII but unable to bind ALK4 and which would be, therefore, candidate type II activin receptor antagonists. Using the structure of ***BMP*** -2 bound to its type I receptor (ALK3) as a guide, we introduced mutations in the context of the inhibin .beta.A cDNA and assessed the signaling activity of the resulting mutant proteins. We identified several mutants in the finger (M91E, I105E, MI0SA) and wrist (activin A/activin C chimera, S60P, 163P) regions of activin-A with reduced signaling activity. Of these the M108A mutant displayed the lowest signaling activity while retaining wild-type-like affinity for ActRII. Unlike wild-type activin-A, the M108A mutant was unable to form a cross-linked complex with ALK4 in the presence of ActRII indicating that its ability to bind ALK4 was disrupted. This data suggested that the M108A mutant might be capable of ***modulating*** signaling of activin and related ligands. Indeed, the M108A mutant antagonized activin-A and ***myostatin*** , but not TGF-.beta., signaling in 293T cells, indicating it may be generally capable of blocking ligands that signal via ActRII/IIB.

L6 ANSWER 26 OF 40 USPATFULL on STN DUPLICATE 3
ACCESSION NUMBER: 2003:219268 USPATFULL <<LOGINID::20060920>>
TITLE: Methods and compositions for the use of stromal cells to support embryonic and adult stem cells
INVENTOR(S): Luft, Christopher, Chapel Hill, NC, UNITED STATES
Wilkison, William O., Bahama, NC, UNITED STATES
Cheatham, Bentley, Durham, NC, UNITED STATES
Gimble, Jeffrey M., Chapel Hill, NC, UNITED STATES
Halvorsen, Yuan-Di C., Branford, CT, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003152558	A1	20030814
APPLICATION INFO.:	US 2002-293394	A1	20021112 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-344555P	20011109 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: KING & SPALDING, 191 PEACHTREE STREET, N.E., ATLANTA,

GA, 30303-1763
NUMBER OF CLAIMS: 37
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 1143
AB The invention provides cells, compositions and methods based on the use of stromal cells to support the proliferation of undifferentiated embryonic or adult stem cells in vitro. The stem cells produced in the method are useful in providing a source of uncommitted or differentiated and functional cells for research, transplantation and development of tissue engineered products for the treatment of human diseases and traumatic tissue injury repair in any tissue or organ site within the body.

L6 ANSWER 27 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2003:200439 USPATFULL <<LOGINID::20060920>>
TITLE: Antibody inhibitors of GDF-8 and uses thereof
INVENTOR(S): Aghajanian, Jane, Belgrade, ME, UNITED STATES
Dunham, William J., Belgrade, ME, UNITED STATES LR
Wolfman, Neil M., Dover, MA, UNITED STATES
O'Hara, Denise, Reading, MA, UNITED STATES
Davies, Monique V., Harpswell, MA, UNITED STATES
Veldman, Geertruida M., Sudbury, MA, UNITED STATES
Bridges, Kristie Grove, Maynard, MA, UNITED STATES
Whittemore, Lisa-Anne, East Walpole, MA, UNITED STATES
Khurana, Tejvir S., Narberth, PA, UNITED STATES
Bouxsein, Mary L., Brookline, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003138422	A1	20030724
APPLICATION INFO.:	US 2002-253532	A1	20020925 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-324528P	20010926 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FINNEGAN, HENDERSON, FARABOW,, GARRETT & DUNNER, L.L.P., 1300 I Street, N.W., Washington, DC, 20005
NUMBER OF CLAIMS: 54
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Page(s)
LINE COUNT: 2606
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The disclosure provides novel antibodies against growth and differentiation factor-8 (GDF-8), including antibody fragments, which inhibit GDF-8 activity in vitro and in vivo. The disclosure also provides methods for diagnosing, preventing, or treating degenerative disorders of muscle, bone, or insulin metabolism.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 28 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2003:152740 USPATFULL <<LOGINID::20060920>>
TITLE: Modified and stabilized GDF propeptides and uses thereof
INVENTOR(S): Wolfman, Neil M., Dover, MA, UNITED STATES
Khor, Soo-Peang, Winchester, MA, UNITED STATES
PATENT ASSIGNEE(S): American Home Products Corporation, Madison, NJ, UNITED STATES, 07054-0874 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003104406	A1	20030605
APPLICATION INFO.:	US 2002-71499	A1	20020208 (10)

	NUMBER	DATE

PRIORITY INFORMATION: US 2001-267509P 20010208 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: c/o Rebecca M. McNeill, FINNEGAN, HENDERSON, FARABOW,, GARRETT & DUNNER, L.L.P., 1300 I Street, N.W., Washington, DC, 20005-3315
NUMBER OF CLAIMS: 118
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 22 Drawing Page(s)
LINE COUNT: 2487
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Modified and stabilized propeptides of Growth Differentiation Factor proteins, such as GDF-8 and Bone Morphogenetic Protein-11, are disclosed. Also disclosed are methods for making and using the modified propeptides to prevent or treat human or animal disorders in which an increase in muscle tissue would be therapeutically beneficial. Such disorders include muscle or neuromuscular disorders (such as amyotrophic lateral sclerosis, muscular dystrophy, muscle atrophy, congestive obstructive pulmonary disease, muscle wasting syndrome, sarcopenia, or cachexia), metabolic diseases or disorders (such as such as type 2 diabetes, noninsulin-dependent diabetes mellitus, hyperglycemia, or obesity), adipose tissue disorders (such as obesity), and bone degenerative diseases (such as osteoporosis).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 29 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2003:314469 USPATFULL <<LOGINID::20060920>>
TITLE: Growth differentiation factor receptors, agonists and antagonists thereof, and methods of using same
INVENTOR(S): Lee, Se-Jin, Baltimore, MD, United States
McPherron, Alexandra C., Baltimore, MD, United States
PATENT ASSIGNEE(S): The Johns Hopkins University School of Medicine, Baltimore, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6656475	B1	20031202
APPLICATION INFO.:	US 2000-626896		20000727 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 485046

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-54461P	19970801 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Eyler, Yvonne
ASSISTANT EXAMINER: Andres, Janet L.
LEGAL REPRESENTATIVE: Gray Cary Ware & Freidenrich, LLP, Haile, Lisa A., Imbra, Richard J.
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT: 6570
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a substantially purified growth differentiation factor (GDF) receptor, including a GDF-8 (myostatin) receptor, as well as functional peptide portions thereof. In addition, the invention provides a virtual representation of a GDF receptor or a functional peptide portion thereof. The present invention also provides a method of modulating an effect of myostatin on a cell by contacting the cell with an agent that affects myostatin signal transduction in the cell. In addition, the invention provides a method of ameliorating the severity of a pathologic condition, which is characterized, at least in part, by an abnormal amount, development or metabolic activity of muscle or adipose tissue in a subject, by modulating myostatin signal transduction in a muscle cell or an adipose tissue cell in the subject. The invention also provides a method of modulating the growth of muscle tissue or adipose tissue in a eukaryotic organism by administering an agent that affects myostatin signal transduction to the organism.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 30 OF 40 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2003-441563 [41] WPIDS
DOC. NO. CPI: C2003-116962
TITLE: New composition comprising an isolated stromal cell capable of supporting the in vitro proliferation and maintenance of stem cells in combination with a stem cell, useful for supporting embryonic and adult stem cells.
DERWENT CLASS: B04 D16
INVENTOR(S): CHEATHAM, B; GIMBLE, J M; HALVORSEN, Y C; LUFT, C; WILKISON, W O; HALVORSEN, Y D C
PATENT ASSIGNEE(S): (CHEA-I) CHEATHAM B; (GIMB-I) GIMBLE J M; (HALV-I) HALVORSEN Y C; (LUFT-I) LUFT C; (WILK-I) WILKISON W O; (ARTE-N) ARTECEL SCI INC
COUNTRY COUNT: 102
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003040346	A2	20030515	(200341)*	EN	33
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
US 2003152558	A1	20030814	(200355)		
EP 1451300	A2	20040901	(200457)	EN	
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR					
AU 2002356935	A1	20030519	(200464)		
JP 2005508393	W	20050331	(200523)		58
BR 2002014029	A	20050419	(200528)		
CZ 2004000695	A3	20050615	(200542)		
CN 1596303	A	20050316	(200567)		
MX 2004004310	A1	20050401	(200571)		
HU 2005000477	A2	20050829	(200580)		
AU 2002356935	A8	20051027	(200624)		
IN 2004000747	P2	20060421	(200634)	EN	
KR 2005044395	A	20050512	(200637)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003040346	A2	WO 2002-US36317	20021112
US 2003152558	A1 Provisional	US 2001-344555P	20011109
		US 2002-293394	20021112
EP 1451300	A2	EP 2002-802903	20021112
		WO 2002-US36317	20021112
AU 2002356935	A1	AU 2002-356935	20021112
JP 2005508393	W	WO 2002-US36317	20021112
		JP 2003-542593	20021112
BR 2002014029	A	BR 2002-14029	20021112
		WO 2002-US36317	20021112
CZ 2004000695	A3	WO 2002-US36317	20021112
		CZ 2004-695	20021112
CN 1596303	A	CN 2002-823582	20021112
MX 2004004310	A1	WO 2002-US36317	20021112
		MX 2004-4310	20040506
HU 2005000477	A2	WO 2002-US36317	20021112
		HU 2005-477	20021112
AU 2002356935	A8	AU 2002-356935	20021112
IN 2004000747	P2	WO 2002-US36317	20021112
		IN 2004-KN747	20040602
KR 2005044395	A	WO 2002-US36317	20021112

KR 2004-707095 20040510

FILING DETAILS:

PATENT NO	KIND	PATENT NO
EP 1451300	A2 Based on	WO 2003040346
AU 2002356935	A1 Based on	WO 2003040346
JP 2005508393	W Based on	WO 2003040346
BR 2002014029	A Based on	WO 2003040346
CZ 2004000695	A3 Based on	WO 2003040346
MX 2004004310	A1 Based on	WO 2003040346
HU 2005000477	A2 Based on	WO 2003040346
AU 2002356935	A8 Based on	WO 2003040346
KR 2005044395	A Based on	WO 2003040346

PRIORITY APPLN. INFO: US 2001-344555P 20011109; US 2002-293394 20021112

AN 2003-441563 [41] WPIDS

AB WO2003040346 A UPAB: 20030630

NOVELTY - A composition comprising an isolated stromal cell capable of supporting the in vitro proliferation and maintenance of stem cells in combination with a stem cell, is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a method for the growth and maintenance of cultured stem cells by isolating tissue-derived stromal cells, and culturing the stromal cells in culture media with stem cells.

USE - The composition and stromal cells are useful for supporting embryonic and adult stem cells, and for the growth and maintenance of cultured stem cells. They are also useful as feeder layers in the isolation, culture and maintenance of adult, embryonic and other stem cells.

Dwg.0/3

L6 ANSWER 31 OF 40 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN DUPLICATE

ACCESSION NUMBER: 2003244765 ESBIOBASE <<LOGINID::20060920>>

TITLE: Myostatin signals through a transforming growth factor .beta.-like signaling pathway to block adipogenesis

AUTHOR: Rebbapragada A.; Benchabane H.; Wrana J.L.; Celeste A.J.; Attisano L.

CORPORATE SOURCE: L. Attisano, Department of Biochemistry, Medical Sciences Bldg., University of Toronto, Toronto, Ont. M5S 1A8, Canada.
E-mail: liliana.attisano@utoronto.ca

SOURCE: Molecular and Cellular Biology, (2003), 23/20 (7230-7242), 65 reference(s)
CODEN: MCEBD4 ISSN: 0270-7306

DOCUMENT TYPE: Journal; Article

COUNTRY: United States

LANGUAGE: English

SUMMARY LANGUAGE: English

AB ***Myostatin*** , a transforming growth factor .beta. (TGF-.beta.) family member, is a potent negative regulator of skeletal muscle growth. In this study we characterized the ***myostatin*** signal transduction pathway and examined its effect on bone morphogenetic protein (***BMP***)-induced adipogenesis. While both ***BMP7*** and ***BMP2*** activated transcription from the ***BMP*** -responsive I-BRE-Lux reporter and induced adipogenic differentiation, ***myostatin*** ***inhibited*** ***BMP7*** - but not ***BMP2*** -mediated responses. To dissect the molecular mechanism of this antagonism, we characterized the ***myostatin*** signal transduction pathway. We showed that ***myostatin*** binds the type II Ser/Thr kinase receptor. ActRIIB, and then partners with a type I receptor, either activin receptor-like kinase 4 (ALK4 or ActRIB) or ALK5 (T.beta.RI), to induce phosphorylation of Smad2/Smad3 and activate a TGF-.beta.-like signaling pathway. We demonstrated that ***myostatin*** prevents ***BMP7*** but not ***BMP2*** binding to its receptors and that ***BMP7*** -induced heteromeric receptor complex formation is blocked by competition for the common type II receptor, ActRIIB. Thus,

our results reveal a strikingly specific antagonism of ***BMP7*** -mediated ***processes*** by ***myostatin*** and suggest that ***myostatin*** is an important regulator of adipogenesis.

L6 ANSWER 32 OF 40 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 5
ACCESSION NUMBER: 2002:676196 CAPLUS <<LOGINID::20060920>>
DOCUMENT NUMBER: 137:212638
TITLE: cDNA and protein sequence of inhibitors of growth differentiation factor-8 (GDF-8) proteins of human and methods for their use
INVENTOR(S): Wolfman, Neil M.; Khor, Soo Peang
PATENT ASSIGNEE(S): Wyeth, John, and Brother Ltd., USA
SOURCE: PCT Int. Appl., 109 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002068650	A2	20020906	WO 2002-US3467	20020208
WO 2002068650	A3	20030821		
WO 2002068650	B1	20040108		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2437218	AA	20020906	CA 2002-2437218	20020208
US 2003104406	A1	20030605	US 2002-71499	20020208
EP 1397492	A2	20040317	EP 2002-709366	20020208
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2005507637	T2	20050324	JP 2002-568744	20020208
BR 2002007110	A	20050816	BR 2002-7110	20020208
NO 2003003456	A	20031006	NO 2003-3456	20030804
ZA 2003006064	A	20060222	ZA 2003-6064	20030806
JP 2006001938	A2	20060105	JP 2005-209321	20050719

PRIORITY APPLN. INFO.: US 2001-267509P P 20010208
JP 2002-568744 A3 20020208
WO 2002-US3467 W 20020208

AB This invention relates to inhibitors of Growth Differentiation Factor-8 (GDF-8) proteins and methods for their use. The cDNA and protein sequence of modified and stabilized propeptides of human Growth Differentiation Factor proteins, such as GDF-8 and Bone Morphogenetic Protein-11, are disclosed. Also disclosed are methods for making and using the modified propeptides to prevent or treat human or animal disorders in which an increase in muscle tissue would be therapeutically beneficial. Such disorders include muscle or neuromuscular disorders (such as amyotrophic lateral sclerosis, muscular dystrophy, muscle atrophy, congestive obstructive pulmonary disease, muscle wasting syndrome, sarcopenia, or cachexia), metabolic diseases or disorders (such as type 2 diabetes, noninsulin-dependent diabetes mellitus, hyperglycemia, or obesity), adipose tissue disorders (such as obesity) and bone degenerative diseases (such as osteoporosis).

L6 ANSWER 33 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2002:301743 USPATFULL <<LOGINID::20060920>>
TITLE: Cystine knot growth factor mutants
INVENTOR(S): Weintraub, Bruce D., Rockville, MD, UNITED STATES
Szkudlinski, Mariusz W., Potomac, MD, UNITED STATES

NUMBER	KIND	DATE

PATENT INFORMATION: US 2002169292 A1 20021114
APPLICATION INFO.: US 2001-813398 A1 20010320 (9)
RELATED APPLN. INFO.: Continuation of Ser. No. WO 1999-US5908, filed on 19 Mar 1999, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	WO 1998-US19772	19980922

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Steven B. Kelber, PIPER, MARBURY RUDNICK & WOLFE, L.L.P., 1200 Nineteenth Street N.W., Washington, DC, 20036-2412
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 13856
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions and methods based on mutant Cystine Knot Growth Factors (CKGFs) comprising amino acid substitutions relative to the wild type hormone/growth factor. Mutated glycoprotein hormones, including thyroid stimulating hormone (TSH) and chorionic gonadotropin (CG) are disclosed as exemplary mutant CKGFs. Mutant TSH heterodimers and hCH heterodimers possessed modified bioactivities, including superagonist activity. Accordingly, the present invention provides methods for using mutant CKGFs, CKGF analogs, fragments, and derivatives thereof for treating or preventing diseases. Pharmaceutical and diagnostic compositions, methods of using mutant TSH heterodimers and TSH analogs with utility for treatment and prevention of metabolic and reproductive diseases are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 34 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2002:281671 USPATFULL <<LOGINID::20060920>>
TITLE: Use of follistatin to increase muscle mass
INVENTOR(S): Lee, Se-Jin, Baltimore, MD, UNITED STATES
McPherron, Alexandra C., Baltimore, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002157126	A1	20021024
	US 6891082	B2	20050510
APPLICATION INFO.:	US 2001-841730	A1	20010424 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-626896, filed on 27 Jul 2000, PENDING Continuation-in-part of Ser. No. US 2000-485046, filed on 5 May 2000, PENDING A 371 of International Ser. No. WO 1998-US15598, filed on 28 Jul 1998, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-54461P	19970801 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Lisa A. Haile, GRAY CARY WARE & FREIDENRICH LLP, Suite 1600, 4365 Executive Drive, San Diego, CA, 92121-2189
NUMBER OF CLAIMS: 42
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 7056
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a substantially purified growth differentiation factor (GDF) receptor, including a GDF-8 (myostatin) receptor, as well as functional peptide portions thereof. In addition, the invention provides a virtual representation of a GDF receptor or a functional peptide portion thereof. The present invention also provides a method of modulating an effect of myostatin on a cell by contacting the cell with an agent that affects myostatin signal transduction in the cell. In addition, the invention provides a method of ameliorating the

severity of a pathologic condition, which is characterized, at least in part, by an abnormal amount, development or metabolic activity of muscle or adipose tissue in a subject, by modulating myostatin signal transduction in a muscle cell or an adipose tissue cell in the subject. The invention also provides a method of modulating the growth of muscle tissue or adipose tissue in a eukaryotic organism by administering an agent that affects myostatin signal transduction to the organism.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 35 OF 40 USPATFULL on STN
ACCESSION NUMBER: 2002:235051 USPATFULL <<LOGINID::20060920>>
TITLE: Use of passive myostatin immunization
INVENTOR(S): El Halawani, Mohamed E., St. Paul, MN, UNITED STATES
You, Seungkwon, Suwon, KOREA, REPUBLIC OF

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002127234	A1	20020912
	US 7037501	B2	20060502
APPLICATION INFO.:	US 2001-754826	A1	20010104 (9)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SCHWEGMAN, LUNDBERG, WOESSNER & KLUTH, P.A., P.O. BOX 2938, MINNEAPOLIS, MN, 55402
NUMBER OF CLAIMS: 28
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 1965
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method to alter the phenotype of animals, e.g., avians, which employs passive and active immunization is provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 36 OF 40 USPATFULL on STN DUPLICATE 6
ACCESSION NUMBER: 2001:163000 USPATFULL <<LOGINID::20060920>>
TITLE: Protein fragment complementation assays for the detection of biological or drug interactions
INVENTOR(S): Michnick, Stephen William Watson, Westmount, Canada
Remy, Ingrid, Montreal, Canada
PATENT ASSIGNEE(S): Odyssey Pharmaceuticals Inc., San Ramon, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6294330	B1	20010925
APPLICATION INFO.:	US 1998-124850		19980730 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1998-17412, filed on 2 Feb 1998

	NUMBER	DATE
PRIORITY INFORMATION:	CA 1997-2196496	19970131

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Brusca, John S.
LEGAL REPRESENTATIVE: Angres, Isaac
NUMBER OF CLAIMS: 64
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Figure(s); 12 Drawing Page(s)
LINE COUNT: 3238
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a general protein-fragment complementation assays to detect biomolecular interactions in vivo and in vitro. The protein-complemetation assay/universal reporter system can be used to detect and screen an agonist and an antagonist of a membrane receptor system. The assay can be used to study protein-protein, protein-DNA, protein-RNA, protein-carbohydrate, and protein-small molecule interactions. The assay can be used to screen cDNA libraries for binding

of a target protein with unknown proteins or libraries of small organic molecules for biological activity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 37 OF 40 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2001-328325 [34] WPIDS
DOC. NO. CPI: C2001-100683
TITLE: Single-chain oligomeric antagonist polypeptide, has one receptor binding site capable of binding and another incapable of binding to ligand-binding domain of receptor and thus binds to receptor but does not activate it.
DERWENT CLASS: B04 D16
INVENTOR(S): ANDERSEN, K V; HALKIER, T; JEPPESEN, C B; NISSEN, T L; OKKELS, J S; SCHAMBYE, H T R; SONI, B; VAN DEN HAZEL, B; SCHAMBYE, H T
PATENT ASSIGNEE(S): (MAXY-N) MAXYGEN APS
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001025277	A1	20010412	(200134)*	EN	123

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW

AU 2000076457	A	20010510	(200143)
EP 1226173	A1	20020731	(200257) EN

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI

US 2004014948	A1	20040122	(200407)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001025277	A1	WO 2000-DK563	20001006
AU 2000076457	A	AU 2000-76457	20001006
EP 1226173	A1	EP 2000-965860	20001006
		WO 2000-DK563	20001006
US 2004014948	A1 Provisional	US 1999-160820P	19991021
	Provisional	US 2000-174655P	20000106
	Provisional	US 2000-225723P	20000816
	Cont of	US 2000-684720	20001006
		US 2003-444691	20030523

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000076457	A Based on	WO 2001025277
EP 1226173	A1 Based on	WO 2001025277

PRIORITY APPLN. INFO: DK 2000-1119 20000720; DK 1999-1438 19991007; DK 1999-1855 19991223
AN 2001-328325 [34] WPIDS
AB WO 200125277 A UPAB: 20010620
NOVELTY - Single-chain oligomeric polypeptide (I) binding to extracellular ligand-binding domain (LD) of cellular receptor (R) which requires binding of oligomeric ligand to 2 or more receptor subunits, to be activated, is new.
DETAILED DESCRIPTION - (I) comprises 3 receptor-binding sites, of which one is capable of binding and the other is incapable of binding to LD of (R). Thus (I) is capable of binding to (R) but incapable of activating (R).
INDEPENDENT CLAIMS are also included for the following:

(1) a nucleotide sequence (II) encoding (I);
(2) an expression vector (III) comprising (II); (3)
(3) a recombinant host cell (IV) comprising (II) or (III);
(4) producing (II) involves mutagenizing a nucleotide sequence encoding a single-chain polypeptide, so as to render at least one receptor-binding site of the polypeptide encoded by the nucleotide sequence incapable of effectively binding to LD of (R);
(5) preparation of (I); and
(6) a pharmaceutical composition comprising (I) together with at least one excipient or vehicle.

ACTIVITY - Antirheumatoid; antiarthritic; antipsoriatic; antiinflammatory; immunosuppressive; dermatological; antibacterial; antiatherosclerotic; cardiant; tranquilizer; immunomodulator; cerebroprotective.

MECHANISM OF ACTION - TNF receptor antagonist. No supporting data is given.

USE - (I) has therapeutic uses and is useful for the preparation of a medicament for the prevention or treatment of a disease or condition involving increased signal transduction from or increased activation of an oligomeric (R). (I), a single-chain trimeric TNF receptor antagonist polypeptide, is useful for the preparation of a medicament for the prevention or treatment of a disease or condition involving undesirable activation of the TNF receptor, or for counteracting undesirable effects of endogenous or exogenous TNF- alpha . (I) is thus useful for preparing a medicament for treating an inflammatory disease or condition such as rheumatoid arthritis, Crohn's disease, systemic lupus erythematosus, Sjogren's disease, cachexia, diabetes mellitus, septic shock, myastenia gravis, juvenile arthritis, atherosclerosis, myocardial infarction, psoriasis, psoriasis arthritis, morbus Still, Wegener's granulomatosis, uveitis, ankylosing spondylitis, acute inflammatory conditions, post-surgical stress, and brain damage (claimed).

ADVANTAGE - With the oligomeric polypeptides comprising at least two or three receptor-binding sites in single-chain form expressed from one continuous nucleotide sequence it is possible to selectively modify at least one receptor-binding site, and leave the other binding site(s) intact through assymetrical mutagenesis. In addition, the single-chain form of the polypeptides more readily lends itself to production by recombinant DNA techniques in that the polypeptides may be expressed from a single gene rather than being assembled in the cell from two or more individual monomers (homomers) or heteromers. Single-chain polypeptides have greater stability upon administration, so that they may exhibit a longer half-life in vivo.

Dwg.0/3

L6 ANSWER 38 OF 40 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2000-387615 [33] WPIDS
DOC. NO. CPI: C2000-117635
TITLE: Administration of a morphogen to treat and alleviate the symptoms of cancer, such as abnormal cell morphology, abnormal enzyme levels opportunistic infection and pain.
DERWENT CLASS: B04 D16
INVENTOR(S): COHEN, M; RUEGER, C; SAMPATH, T; COHEN, C M; RUEGER, D C; SAMPATH, K T
PATENT ASSIGNEE(S): (CREA-N) CREATIVE BIOMOLECULES INC; (CURI-N) CURIS INC
COUNTRY COUNT: 90
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000029012	A2	20000525	(200033)*	EN	75
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000016167	A	20000605	(200042)		
EP 1131087	A2	20010912	(200155)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					

JP 2002529513 W 20020910 (200274) 83
EP 1435243 A2 20040707 (200444) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
EP 1131087 B1 20040908 (200459) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
DE 69920033 E 20041014 (200468)
ES 2226467 T3 20050316 (200525)
DE 69920033 T2 20050922 (200562)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000029012	A2	WO 1999-US26636	19991112
AU 2000016167	A	AU 2000-16167	19991112
EP 1131087	A2	EP 1999-958892	19991112
		WO 1999-US26636	19991112
JP 2002529513	W	WO 1999-US26636	19991112
		JP 2000-582058	19991112
EP 1435243	A2 Div ex	EP 1999-958892	19991112
		EP 2004-8347	19991112
EP 1131087	B1	EP 1999-958892	19991112
		WO 1999-US26636	19991112
	Related to	EP 2004-8347	19991112
DE 69920033	E	DE 1999-620033	19991112
		EP 1999-958892	19991112
		WO 1999-US26636	19991112
ES 2226467	T3	EP 1999-958892	19991112
DE 69920033	T2	DE 1999-620033	19991112
		EP 1999-958892	19991112
		WO 1999-US26636	19991112

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000016167	A Based on	WO 2000029012
EP 1131087	A2 Based on	WO 2000029012
JP 2002529513	W Based on	WO 2000029012
EP 1435243	A2 Div ex	EP 1131087
EP 1131087	B1 Related to	EP 1435243
	Based on	WO 2000029012
DE 69920033	E Based on	EP 1131087
	Based on	WO 2000029012
ES 2226467	T3 Based on	EP 1131087
DE 69920033	T2 Based on	EP 1131087
	Based on	WO 2000029012

PRIORITY APPLN. INFO: US 1998-191239 19981113
AN 2000-387615 [33] WPIDS
AB WO 200029012 A UPAB: 20000712
NOVELTY - Use of a morphogen to treat or alleviate the symptoms of cancer, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:
(1) a method for alleviating the symptoms of cancer comprising administering a morphogen which inhibits growth of cancer cells and comprises a dimeric protein with an amino acid (aa) sequence:
(a) having at least 70% aa homology with the C-terminal seven cysteine skeleton of osteogenic protein (OP)-1, residues 330-341 of (I) which has a defined sequence of 431 aa given in the specification; or
(b) having at least 60% aa sequence identity with the C-terminal seven cysteine skeleton of OP-1;
(2) a method for reducing the number of cancer cells in a population of cancer cells comprising administering a morphogen which inhibits cellular incorporation of (3H)-thymidine in an in vitro assay and comprises a dimeric protein with an amino acid sequence:
(a) having at least 70% aa homology with the C-terminal seven cysteine skeleton of OP-1, residues 330-341 of (I);
(b) having at least 60% aa sequence identity with the C-terminal

seven cysteine skeleton of OP-1; or

(c) defined by sequences (II)-(VI) of 97 or 102 aa given in the specification;

(3) a method for alleviating the symptoms of cancer comprising administering a bone morphogenic protein (BMP), or morphogenically active amino acid variants of BMP, particularly a naturally occurring allelic variant, a conservative aa substitution variant, a peptide fragment variant or a conservative aa substitution variant of the peptide fragment variant of BMP; and

(4) a method of treating cancer in a mammal comprising determining whether targeted cancer cells in the mammal express at least one bone morphogenic protein receptor (BMPR) and administering a morphogen specific for the BMPR which inhibits growth of cancer cells and comprises a dimeric protein with an aa sequence:

(a) having at least 70% aa homology with the C-terminal seven cysteine skeleton of OP-1, residues 330-341 of (I); or

(b) having at least 60% aa sequence identity with the C-terminal seven cysteine skeleton of OP-1.

ACTIVITY - Cytostatic.

Human embryonal carcinoma (EC) cell line NTERA-2CL derived from a human teratocarcinoma were induced to grow in serum free media (SFM). In the absence of morphogen treatment the cells proliferated rampantly and were anchorage-independent. In the presence of osteogenic protein (OP)-1 the EC cells grew as flattened cells, becoming anchorage dependent and the growth rate was reduced 10-fold.

MECHANISM OF ACTION - Gene therapy.

USE - The cancer to be treated is germ cell tumors, leukemia, lymphoma, teratocarcinoma, adrenal, anus, bladder, bone, brain, breast, cervix, colon, corpus, endocrine, esophageal, fallopian tube, fat cell, gall bladder, gastrointestinal tract, kidney, liver, lung, muscle, nervous system, ocular tissue, oral, ovarian, pancreatic, prostate, rectal, skin, small intestine, soft tissue, stomach, testicular, thyroid, ureteral, urinary, uterine or metastatic cancer of unknown primary site (claimed). Symptoms which are alleviated include abnormal bodily function, abnormal cell morphology, abnormal enzyme levels, abnormal hormone levels, abnormal oncofetal levels, abnormal tissue growth, abnormal tissue mass, abnormal tumor-associated protein levels, altered neurologic function, altered neurologic structure, angiogenesis, bleeding, cells with a cancer cell phenotype, diarrhea, effusions, fatigue, fever, lesions, malnutrition, metastasis, nausea, obstruction of a bodily passageway, opportunistic infection, pain, poor Karnofsky performance status, presence of cell-surface markers, presence of histological makers, presence of intracellular markers, presence of molecular markers, tumor invasion, unregulated cell proliferation, urinary frequency and weight loss.

The morphogen reduces tumor size or development, results in tumor necrosis and diffuse inflammation and increases survival rate.

Dwg.0/28

L6 ANSWER 39 OF 40 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 7
ACCESSION NUMBER: 1999:594973 CAPLUS <<LOGINID::20060920>>
DOCUMENT NUMBER: 131:223973
TITLE: Use of follistatin to modulate GDF-8 and BMP-11 and treat diseases associated with the two proteins A
INVENTOR(S): Wood, Clive R.; Fitz, Lori Jo
PATENT ASSIGNEE(S): Genetics Institute, Inc., USA
SOURCE: PCT Int. Appl., 15 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9945949	A2	19990916	WO 1999-US4003	19990224
WO 9945949	A3	19991118		

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT,

UA, UG, UZ, VN, YU, ZW
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6004937	A	19991221	US 1998-37118	19980309
CA 2322716	AA	19990916	CA 1999-2322716	19990224
AU 9927854	A1	19990927	AU 1999-27854	19990224
AU 759838	B2	20030501		
EP 1061940	A2	20001227	EP 1999-908416	19990224
EP 1061940	B1	20031217		

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

JP 2002506044	T2	20020226	JP 2000-535362	19990224
AT 256475	E	20040115	AT 1999-908416	19990224
PT 1061940	T	20040331	PT 1999-908416	19990224
ES 2212533	T3	20040716	ES 1999-908416	19990224
EP 1444985	A2	20040811	EP 2003-28901	19990224

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, MK, CY, AL

AU 2003231603	A1	20030911	AU 2003-231603	20030801

PRIORITY APPLN. INFO.: US 1998-37118 A 19980309
AU 1999-27854 A3 19990224
EP 1999-908416 A3 19990224
WO 1999-US4003 W 19990224

AB The present invention provides ***methods*** for ***modulating*** the effects on cells of growth and differentiation factor 8 [***GDF*** - ***8***] and bone morphogenetic protein 11 [***BMP*** -11] by administering follistatin to the cells. The invention further provides methods for blocking the effects on cells of GDF-8 or BMP-11 and methods for treating disorders assocd. with neural or muscular effects of GDF-8 or BMP-11 by administering an effective amt. of follistatin.

L6 ANSWER 40 OF 40 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 8
ACCESSION NUMBER: 2000:290953 BIOSIS <<LOGINID::20060920>>
DOCUMENT NUMBER: PREV200000290953
TITLE: Use of follistatin to modulate growth and differentiation factor 8 [GDF-8] and bone morphogenic protein 11 [BMP-11].
AUTHOR(S): Wood, Clive R. [Inventor, Reprint author]; Fitz, Lori Jo [Inventor]
CORPORATE SOURCE: Boston, MA, USA
ASSIGNEE: Genetics Institute, Inc.
PATENT INFORMATION: US 6004937 19991221
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Dec. 21, 1999) Vol. 1229, No. 3. e-file.
CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Jul 2000
Last Updated on STN: 7 Jan 2002

AB ***Methods*** are provided for the ***modulation*** of the effects of ***GDF*** - ***8*** and ***BMP*** -11, particularly on neural and muscular disorders administration of follistatin for treating neural, muscle, disorders which are characterized by an abnormality in the levels or activity of ***GDF*** - ***8*** or ***BMP*** -11.

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L1 QUE (PROMYOSTATIN OR MYOSTATIN OR PRO-MYOSTATIN OR GDF-8)

L2 3512 S L1
L3 1855 S (MODULAT? OR INHIBIT? OR ALTER? OR DECREAS? OR INCREAS?) (S)
L4 148 S (BMP? OR BMP-1 OR (BONE (W) MORPHOGENIC (W) PROTEIN))(S) L3
L5 50 S (METHOD? OR PROCESS?)(S) L4
L6 40 DUP REM L5 (10 DUPLICATES REMOVED)

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